THERMAL BURNS

by
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I have been asked to discuss the modern treatment of burns. It seems to me wise before beginning with the treatment of burns to examine briefly what we know about the pathogenesis of burns, and the cause of death from burns.

Previous to 1925 it was almost universally held that the cause of death from burns was the absorption of various toxic protein split products arising in the burned area as a result of the destruction of large amounts of tissue. About ten years ago that view gave way to the concept that death from burns was due to the large loss of plasma from the circulating blood, with resultant decreased output of the heart, lowered blood pressure and generalized tissue anoxia. Within recent years the concept has arisen that both factors may play a role in the cause of death in burns; that there may be an extensive loss of plasma from the vessels and in addition the absorption of poisons from the burned area. The problem is still unsettled, and I should like to indicate briefly the evidence that we have, upon which we can base a tentative opinion.

First, with respect to the pathology in burns, the local pathology varies all the way from the erythema of the first degree burn, the blistering and vesicle formation of the second degree burn, to the extensive eschar with destruction of the skin and the subcutaneous tissues in the third degree burn. It is important, I think, from the standpoint of treatment to realize that we cannot at the time of first seeing the burn make a sharp diagnosis or differentiation of the extent of the burn or determine the involvement of the deeper tissues. We cannot always distinguish between a second and a third degree burn. We know that the burn is of the third degree when subsequently we find that we have skin grafting to do. This is unfortunate, because if we know the extent of the burn at the time when it was first seen, more successful treatment might be devised.

Perhaps the most important local change is the extensive edema in the burned area and the very large accumulation of plasma or plasma-like fluid in the subcutaneous tissues, and the very large loss of plasma through weeping from the burned surface. Besides this we may find

extensive charring or necrosis of the subcutaneous tissue also involving the muscles and even the bones in very severe burns.

In addition to these local effects, certain systemic changes have been found to occur fairly constantly in severe burns. First I should like to call your attention to the changes which have been described in the liver. Areas of focal necrosis and fatty degeneration are not infrequently found. It is significant that some recent work has demonstrated that tannic acid applied locally or injected beneath the skin can produce focal necrosis and fatty infiltration in the liver, so that it may possibly be that in some cases the focal necrosis and changes in the liver seen in burns treated within the last 12 years may be due not to the burn but to the use of tannic acid. It is important, however, to realize that this change in the liver was described before the advent of tannic acid treatment, and has been found in burned patients who were not given the tannic acid. The cause of this fatty necrosis and fatty change in the liver in burns in not entirely known.

The kidneys in many cases are found to display acute glomerulitis, hemorrhage in the glomeruli and degenerative changes in the epithelium. The gastrointestinal tract very often displays widespread hyperemia, superficial ulceration in some cases, and fairly deep penetrating ulceration in the duodenum occasionally in the pyloric part of the stomach or in the first part of the jejunum. This ulceration in the duodenum was first described by James Long in 1840 and subsequently by Curling in 1842 and as you know is commonly called the Curling ulcer. The pathogenesis of this ulcer is unknown, but I might say to you that an attractive hypothesis suggested to account for it is based on some recent work of Wangensteen and his collaborators at the University of Minnesota. These men have found that the inplantation of histamine in beeswax beneath the skin of experimental animals produces long continued secretion of gastric juice by the stomach. This is due to the gradual liberation and absorption of histamine from the beeswax. Excessive secretion of gastric juice produces ulcers in the stomach or first part of the duodenum. There is some evidence that among the products which may be absorbed from a burned area is histamine and there have been a number of studies indicating that the level of the histamine in the blood in burns is slightly increased. It is conceivable then that the chronic absorption of histamine from the burned site may well provoke a continuous secretion of gastric juice and the formation of

ulcers in the stomach or duodenum. This is, however, not a settled question. I would like, however, to point out that it is difficult to account for the ulcer in the intestines in burns on the basis simply of plasma loss. The adrenals often display hyperemia and hemorrhage. The significance of this is not clear. We find similar hyperemia and hemorrhage following intravenous administration of many types of tissue extracts containing various protein split products. It is now felt, however, that the most significant abnormality in burns is the very large loss of plasma, resulting in extensive hemoconcentration. The proportion of corpuscles to plasma rises from a normal of 44 or 45 per cent to as high as 65 or 70 per cent of the blood volume. There is thus a very large loss of the liquid part of the blood. A fall in the concentration of chlorides in the blood has also been found. This was demonstrated first by Underhill and his associates a number of years ago.

An increase in the clotting time of the blood also occurs, the mechanism of which is at present entirely obscure. It is not due to any alteration in the blood calcium, nor to any alteration in the fibrinogen of the blood or to its prothrombin content. A marked edema in the lungs and in the viscera has also been found.

One view of the pathogenesis of burns holds that death is due to the escape of plasma from the vascular space through weeping from the burned surface, the so called white hemorrhage, accumulation of plasma in the subcutaneous tissue at the burned site, and escape of plasma through the capillaries in the lungs, liver and gastrointestinal tract. Realization of the importance of the loss of the liquid constituents of the blood in burns came about first through the work of Underhill and his associates in 1923. These men reported that a marked hemoconcentration occurred in burns, the hematocrit rising from the normal of 44 to as high as 65 or 75 per cent. A superficial burn in rabbits on which their work was chiefly done involving one-sixth of the body surface caused a loss of 70 per cent of the total blood volume as subcutaneous edema.

Now it is important to know from the standpoint of treatment at least that this escape of plasma from the vascular space occurs with considerable rapidity. In tipping experiments where an animal was placed on a balanced table and a burn applied to one side of the body, accumulation of subcutaneous edema on the burned side of the body was manifested by tipping of the body toward the burned side, and this tipping was found to begin within a few minutes after the application of the burn. Others have found,

and we have confirmed this in our own experiments, that significant loss of plasma is well marked within 15 or 20 minutes from the onset of the burn and may reach its maximum within four to six hours.

It is important then for the physician to realize that in most cases a very marked loss of plasma from the vascular space has already occurred by the time the patient comes to the physician. We had the opportunity to take care of a patient in our clinic sometime ago whom we saw within 30 minutes after the receipt of the burns. The hematocrit was already above 50 and by the time the local treatment had been applied, which took 40 minutes, it rose to over 60, so that the striking rapidity of the loss of plasma from the circulation is apparent. This is important to realize when we come to discuss the rationale of the pressure treatment of burns.

This large loss of plasma into the burned area with resultant decrease in the volume of the circulating blood brings about a fall in blood pressure and decreased cardiac output. The fall in blood pressure may go beyond that which can be compensated for by vasoconstriction, increase in the heart rate, the normal regulatory mechanism. There is some evidence that local tissue asphyxia resulting from long continued low blood pressure may produce a widespread capillary damage so that plasma escapes from the vessels not only in the burned area but also in distant organs such as the lungs, liver, or gastrointestinal tract.

This theory then attempts to account for the pathogenesis of burns not through the absorption of a toxic substance from the burned area, but from the loss of plasma from the vascualr spaces, primarily into the burned site as weeping from the surface and subcutaneous accumulation of plasma, and also to the escape of plasma into the lungs, gastrointestinal tract and visceral organs.

The other point of view holds that death from burns is due to a toxemia due to poisons absorbed from the burned area which enter the circulation, produce widespread capillary damage and increased permeability, damage to the liver, kidneys, adrenal glands and gastrointestinal mucosa. There is now no dispute about the dangerous diminution in the blood volume and hemoconcentration in burns. The evidence upon which the intoxication theory of burns is based is not very firm. I am not going to analyze the earlier experiments in this connection, but refer to the work of Robertson and Boyd in 1923. These workers reported that extracts of burned skin and the blood of

burned animals was toxic. These experiments, however, were repeated by Underhill and his associates, by Blalock, by Harkins, and a number of others, and the results are contradictory. Some have reported increased toxicity, others no increase in toxicity, so that a final decision cannot be made at present. A significant contribution was made by Elkington and Roberts in 1940. These investigators reported that patients with severe burns may die even though the plasma loss and hemoconcentration present has been entirely corrected by the administration of plasma, indicating that some residuary defect exists even when the plasma that has been lost is replaced. I think this is a significant finding. I have no reason to doubt that the experiments indicating a slight increase in the amount of histamine in the blood are also valid. The significance of that finding, however, is difficult to assess. Whether there is sufficient liberation of histamine to produce a significant systemic effect is hard to say. The decreased coagulability of the blood however is a factor suggesting some systemic change. This decreased coagulability of the blood is apparently not due to the presence of some heparine-like substance in the blood.

I may state this experiment to you for what it is worth. We asked ourselves the question, is there absorbed from the burned area a sufficient amount of toxic material to seriously affect the animal? To get some data on this point we arranged for cross circulation between two animals, A and B. In this type of experiment where a large loss of plasma occurs in one of the animals, it is necessary to arrange for quantitative cross circulation, otherwise the animal who is burned will receive all of the blood from the normal animal and the normal animal will bleed to death into the veins of the animal whose blood pressure falls. This was controlled by arranging for a quantitative cross circulation. When the cross circulation was established one of the animals of the pair was severely burned and continuous cross circulation was maintained between the pair. The burned animal finally died at the end of four or five hours. The experiment was repeated a number of times. During the course of the burning the normal animal which was receiving the blood from the burned animal received as much as 3000 c.c., that is, all of the blood from the burned animal was circulated through the body of the normal animal four or five times. When the burned animal was moribund the animals were separated, and in each case we found that the animal which continuously received the blood from the burned animal survived, indicating that so far as one can judge from this type

of experiment a sufficient amount of toxic material to cause death was not elaborated into the circulating blood. Offhand this sounds like a conclusive type of experiment. It has, however, certain limitations. If one, for instance, arranges for a similar cross circulation and then poisons one animal with such a substance as strychnine, giving animal A, let us say, a double lethal dose of strychnine, wait for a minute or two for the strychnine to become distributed throughout the body, and then establishes cross circulation and maintains it continuously, the animal which has been poisoned with strychnine develops typical convulsions and goes on and dies, whereas the other animal which is constantly receiving blood from the strychninized animal at the same time shows no signs whatever of strychnine poisoning. So we know that one of the characteristics of an active toxic substance is that it becomes rapidly fixed in the tissues and does not exist for any long period of time in the circulating blood.

We may take it, I think, that when a burned animal or man dies he does not have more than a lethal dose of toxic material in his entire organism. The chances are good that most of that is fixed in the tissues and that very little of it is in the circulating blood. I point that out simply to indicate the very great difficulty of determining the presence or absence of a toxemai by examining the blood itself.

I am unable to say now what the cause of death in burns is. My provisional feeling is that the most important factor responsible for the death of a burned patient within the first 24 to 48 hours is the large loss of plasma from the vascular space. I believe that it is the principal cause of death and for the symptoms that occur during the first 24 or 48 hours. I believe, however, that it is probable that a certain amount of toxic material enters the circulation from the burned area, and that this toxic material produces widespread effects on distant organs, of which the ulcer in the gastrointestinal tract, the focal necrosis in the liver and diminished coagulability of the blood may be evidence.

Now with respect to the treatment of burns. It seems to me that the principles involved in the treatment of burns are these: to relieve pain, replace the lost plasma, to restore kidney function, to protect the injured tissues, to prevent infection, to limit plasma loss, and to favor repair.

Now, all are agreed that it is safe and useful to relieve the pain of burns by the use of morphine. The OCD recommends the administration of a half grain of morphine and in our experience that has been satisfactory. Most writers caution against the use of a general anesthetic. A general anesthetic is unneeessary in adults, but I must confess in handling children I have felt that there was more advantage to the use of a general anesthetic than danger. However mostly we will have to do, I presume, with burns in adults, and I think pain can be adequately controlled by the administration of this relatively large amount of morphine.

Second, replace the lost plasma. There is no debate whatever concerning the presence of marked hemoconcentration in burns. All also recognize that very large amounts of plasma are necessary to restore the blood to its normal state. There is some disagreement concerning the wisdom of trying to replenish the lost plasma immediately be a single transfusion if plasma is available, or whether it is not better to administer the plasma required in fractional doses. A large amount of plasma is required. A burn that involves 10 per cent of the body ordinarily will require a thousand c.c. of plasma to restore the concentration of blood to its normal level. I should like to point out, however, that the loss of plasma begins immediately after the receipt of the burn, becomes very marked within a few hours and continues for three or four days. Attempts have been made to devise a formula indicating the amount of plasma which ought to be given in an attempt to completely replace the loss of plasma by the time the patient is seen. I doubt that that is necessary. I think it is probably better to give the patient perhaps 500 c.c. of plasma when he is first seen, realizing that if the hematocrit is 70 that may bring the hematocrit down to only 65 or 60, then four or five or six hours later a second administration of plasma repeated again in five or six hours. I believe that is more beneficial than the administration of the entire volume of 1500 or 2000 cc. when the patient is first seen. That large amount of plasma is also often not available. The point I want to bring out is there is some experimental evidence indicating that the stage administration of the plasma and gradual correction of the hemoconcentration has some advantage.

Restoring kidney function. Many of these patients have a diminished urinary secretion or a complete anuria. That was evident in four patients that I saw a year or so ago. We gave these patients a relatively large volume of physiological salt solution intravenously. I believe

one ought to give these patients as much physiological salt solution intravenously as one administers plasma. There is this objection to the administration of physiological salt solution: in the presence of damaged capillaries it passes rapidly into the subcutaneous tissue, and some authors have suggested that it not only passes out itself, but it carries plasma along with it. I doubt that, however. It does pass into the subcutaneous tissue and to a certain extent increases the edema, but it is very effective in restoring urinary flow, and in view of the evidence that we have, not good to be sure, but suggestive evidence, that a toxemia of some type may exist, I believe it is wise to correct the anuria that exists in severe burns, even at the risk of increasing the local edema.

In burns there is always more or less hemolysis of the blood, destruction of corpuscles resulting in staining of the plasma with hemoglobin. Whether this is due to the destruction of the corpuscles locally by the heat or whether it is due to a toxemia is not known, but a moderate degree of anemia may be looked for in severe burns, even if infection is avoided, and particularly so if infection occurs. So that blood transfusion is almost always indicated in severe burns.

Now, the local treatment of burns. The efforts here are to protect the injured tissues, to prevent infection, to limit plasma loss, and to favor repair. All are agreed that the prevention of infection is the most important single factor in the local treatment of burns. The organisms that are commonly present in the skin are usually not serious. The serious organisms that infect burns are usually deposited there from the nose and throat of the physician or attendant who first sees the burn. This means that the man who takes care of the burn must exercise the same aseptic precautions that he exercises in a laparotomy; i.e., rubber gloves, mask, gown, and sterile technique. If a burn is seen shortly after it is received, it is likely that the burned area is sterile. If it has not been contaminated by organisms from the nose and throat of the patient or from the first aid attendants, it is likely that it could be successfully handled without any attempt to clean the wound. It is interesting to note, in the Cocoanut Grove disaster, results in some of the patients brought to the Massachusetts General Hospital. Here the wounds were simply covered with sterile dressing when the patients were first received and then a bland mild antiseptic ointment and a pressure dressing were applied. This sufficed to prevent any serious infection in a large number of the

patients so treated.

If the burn however is dirty, the tissues are covered with oil or dirt or foreign bodies, I believe it is wise to adopt the technique recommended by Sumner Koch of Chicago, that is, careful cleansing of the wound with sterile water and soap, generally be means of cotton pledgets. He has demonstrated that it is possible to mechanically convert an infected wound into a sterile wound by washing with soap and water. It is probably wise also to remove the dead skin around blisters and cut away all dead or devitalized tissues.

Now, we come to a question about which there is a difference of opinion. In 1925, E. C. Davidson, introduced the tannic acid treatment of burns, and there can be no question that this was at the time of a great advance. At this period the significance of the loss of plasma in the pathogenesis of burns was not fully appreciated. The application of tannic acid limits the local loss of plasma, because it provides a tough tanned membrane which stops weeping and provides a certain amount of local compression. The rational of the tannic acid treatment as introduced by Davidson was curiously enough that the tannic acid by tanning the burned area limits the absorption of poisons. That was the theory back of the treatment.

Irrespective of that, it must be conceded that the introduction of the tannic acid treatment of burns saved a great many lives. Why then do people object to the tannic acid treatment of burns at the present time? The objections are based largely on these points:

With the recognition of the significance of the loss of plasma and the availability of plasma for infusion, the necessity of using tannic acid for that purpose is not so important as it was at first.

Second, it is altogether probable that tannic acid destroys a certain amount of surviving epithelium in a burned area. It coagulates not only dead protein, but it destroys many islands of surviving epithelium in the depths of hair follicles from which island regenerating skin might otherwise cover the burned site.

I have no doubt from my own experience that tannic acid often converts a second degree burn into a third degree burn. Of greater significance, however, is the point that the tannic acid coagulum over a burned area handicaps the surgeon in the treatment of infection. It is likely that

if one could be sure that all burns were aseptic or could be made so, most of the objection to the tannic acid treatment would vanish, but in my experience this does not obtain. In very large burns, in spite of a reasonably rigorous attempt to wash the wound clean, and a certain amoung of judgment has to be exhibited here for fear of damaging the tissue, a variable number of organisms persist. If a dense coagulum is completely over the burned area, the presence of an early infection is difficult to detect, pus may accumulate beneath the membrane and a large part of the wound may become seriously infected before it is realized.

That is the first objection that I have to the tannic acid treatment.

Another point comes in the demonstration of the toxic effect of tannic acid on the liver. There seems no doubt that tannic acid itself can produce focal necrosis and fatty degeneration of the liver. Whether this happens in any large number of burn cases treated with tannic acid is not possible to say, but I believe that it is a point that ought to be kept in mind.

An alternative is illustrated by a method of treatment popularized by Sumner Koch and Allen and reported in 1942. With a large series of burns Koch and Allen demonstrated that it was possible to secure comparatively aseptic wounds by washing with sterile water and soap, with an aseptic technique.

After the wound is cleansed, vaseline gauze and a protective dressing is immediately applied and a pressure dressing. This is done with the aid of sponges, with gauze, with the application of a roller or an elastic bandage.

The thought behind the pressure dressing is that the pressure dressing limits the continued escape of plasma from the burned area, by the application of mechanical pressure. It also immobilizes the limb and the injured joints quite effectively, almost as effectively as a plaster cast, and contributes thereby greatly to the comfort of the patient.

We have used this method of Koch and Allen, and decided that it represents a real advance. We have added to it one point. Instead of using vaseline gauze as a protective dressing we have made use of the specific bacteriostatic action of the sulfonamides. The sulfonamide that we have preferred is sulfathiazole, because it is more versatile in its action against organisms than the other sulfonamides,

and it also seems to have an analgesic action.

Dr. J. Garrott Allen of our clinic prepared sulfathiazole 20 per cent in an ointment base, and we have used this on gauze in place of the vaseline of the Koch and Allen pressure dressing method.

I believe this represents the best method of treatment of burns at the present time; debridement, cleansing, the application of an ointment protective dressing, which may well be one of the sulfonamides, and the pressure dressing.